VALIDITY OF COLD IRRIGATION TEST (CIT) USED IN TERTIARY CARE FOR DIAGNOSING VESTIBULAR DYSFUNCTION

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A dissertation submitted in partial fulfillment of the rules and regulation for the **M.S. (Branch IV) Otorhinolaryngology** examination of the **Tamil Nadu Dr. M.G.R Medical University** to be held in **March 2008.**

CERTIFICATE

This is to certify that the dissertation entitled "**Validity of cold irrigation test (CIT) used in tertiary care for diagnosing vestibular dysfunction"** is a bonafide original work of Dr. Ramesh Menon U submitted in partial fulfillment of the rules and regulation for the **M.S. (Branch IV) Otorhinolaryngology** examination of the **Tamil Nadu Dr. M.G.R Medical University** to be held in **March 2008.**

Dr. Rupa Vedantam MS., DLO
Professor & Head
Dept. of ENT
Christian Medical College & Hospital
Vellore – 632 004.

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Dr. Achamma Balraj MS., DLO
Professor & Guide
Dept. of ENT
Christian Medical College & Hospital
Vellore – 632 004.

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. . . Dr. U. Ramesh Menon

CONTENTS

INTRODUCTION

Disorders of balance cause the patient to present in different clinics like ENT, cardiology, neurology and geriatric medicine, and thus different specialities develop different protocols for evaluation and treatment in their own areas of expertise. Thus focused ways of treatment while having some advantages often cause doctors to overlook signs and symptoms other than their own speciality and further causes problems like unnecessary expensive investigations being done. This puts a strain on the patient's scarce resources in developing countries and also burdens health care systems in developed countries that have to bear the cost of repeated referrals and expensive investigations often with no clear diagnosis.

A disorder of balance also puts severe restrictions on patient's ability to work and the patient risks loosing his job due to continuous absence is estimated that patients with balance disorders constitutes nearly twenty percent of the patient load in an ENT clinic (Luxon 2002).

Minor pathologies in vestibular system can cause major disability. Hence management strategies should include more of disability and handicap alleviation rather than on pathology alone.

The purpose of this study is to evaluate the validity of a screening test which is simple and cost effective for diagnosis of vertigo and compare it with available gold standard test in order to determine if the screening test has sufficient sensitivity and specificity to be retained in a tertiary centre.

AIMS AND OBJECTIVES

Aim

To find out if cold irrigation test can be used as a screening test to identify patients with abnormality of the vestibular system.

Null hypothesis:

Cold irrigation test can be used as a screening test to detect abnormality of the vestibular system in patients with symptomatic vertigo

Objectives

1) Describe the range of values from cold caloric test in normal persons (asymptomatic)
2) Describe the range of values from cold caloric test among symptomatic patients
3) Assess the suitability of cold caloric test for screening in symptomatic patients for further tests

REVIEW OF LITERATURE

DEVELOPMENT OF THE INNER EAR:

At about the 22 to 23 day of gestation when the human embryo reaches the seventh somite stage an ectodermal thickening forms close to the neural tube that forms the brain and cranial nerves. This thickening is the otic plocade that deepens and sinks below the surface to form the otic pit which later forms the otocyst. Associated with the otocyst are neural crest cells that later separate into facial (Geniculate), auditory (Spiral) and vestibular (Scarpas) ganglions. A series of spectacular changes in the otocyst result in the adult membranous labyrinth at 25 weeks of gestation (Glasscock Gulya 2003).

Within one to two days of the formation of the otic cyst, two divisions the endolymphatic division and the utriculosaccular division become visible, the former gives rise to the utricle and the semicircular canal and latter to the saccule and cochlea.

The semicircular canal develops at around 35 days and the superior semicircular canal is the first to develop by about six weeks.

The sensory cells of the three cristae and two maculae develop around seven weeks of gestation (Lysakowski 2005). The hair cells of the vestibular end organs are fully developed by the ninth week. The maculae of otoliths are fully developed by about the 14 to 16 weeks; cristae of the semicircular canal by about 23 weeks and organ of Corti by about the 15 weeks (Gulya 2003).

The bony labyrinth is formed by the chondrification of the mesenchyme surrounding the otocyst. Dedifferentiation takes place to form the perilymphatic space. A communication forms with the cerebrospinal fluid through the cochlear aqueduct that runs to the posterior cranial fosse.

The membranous labyrinth is housed within the bony labyrinth in the petrous portion of the temporal bone (Figure 1), where it is secured by connective tissue and is bathed in perilymph. Endolymph is contained within the membranous structure, where the specialized sensory neuro-epithelium is located. The vestibular apparatus consists of two groups of specialized sensory receptors.

Three semicircular canals – lateral, posterior, and superior, each of which originates from the utricle and terminates in a dilated, end (ampulla) that also opens into utricle.

Two otolith organs – the utricular and the saccular macula, are located in the vestibule.

The semicircular canals are oriented in approximately orthogonal planes to the other ipsilateral canals. Although the two horizontal canals are in parallel planes, the two superior and the posterior canals are in planes approximately orthogonal to each other. The canals are organized into functional pairs. The two members of each pair are in parallel planes of orientation.

The otolith organs also function in a paired format, with the two utricular maculae in approximately the horizontal plane and the two saccular maculae in the vertical plane, with an approximate 30^0 angulation inward to the midsagittal plane.

Within the semicircular canal ampulla and otolith organ there are specialized hair cells which constitute the neuroepithelial transduction mechanism for the vestibular end organs. There are two sense organs in the vestibular labyrinth: the crista ampullaris in the semicircular canals, which respond to the angular acceleration (head turning), and the maculae of the utricle and saccule, which respond to linear acceleration (up and down forward and backward, and sideways movement).

Figure 1: The orientation of the labyrinths in the temporal bone

The crista ampullaris is shaped like a small hill and contains hair cells from which protrude many stereocillia and a single kinocilium embedded in a gelatinous substance called the cupula. The kinocilium in the horizontal semicircular canal is located towards the utricle and the stereocilia are away from the utricle. In the posterior and superior semicircular canals, however, the kinocilium is located away and the stereocilia are located towards the utricle (Figure 2)

Deflection of the kinocilium toward the utricle in horizontal, superior, and posterior canals result in utricolpetal (ampulopetal) deviation. Utriculopetal deviation is associated with increased electrical activity in the horizontal semicircular canal and decreased electrical activity in the superior and posterior semicircular canals. Deflection of kinocilium away from the utricle in the horizontal, superior and posterior canals results in utricofugal (ampullofugal) deviation. This deviation is associated with decreased electrical output in the horizontal semicircular canal and increased electrical activity in superior and posterior semicircular canals (Figure 3)

The maculae have hair cells and supporting cells. The stereocillia of the hair cells are embedded in a gelatinous material. On the surface of the gelatinous layer are otoconia, composed of calcium carbonate, which transmit the effects of gravity to the underlying hair cells, thereby making the hair cells more sensitive to linear acceleration.

The vestibular branch of the vestibulocochlear nerve innervates the five vestibular sense organs: the cristae ampullaris of the three semicircular canals and the maculae of utricle and saccule. The first order vestibular neurons that are bipolar and situated in the Scarpa's ganglion terminate in the vestibular nuclei in the lower brainstem (figure 4).

Cerebellum, which coordinates reflex and other muscular and neural activity required for orientation (Figure 5)

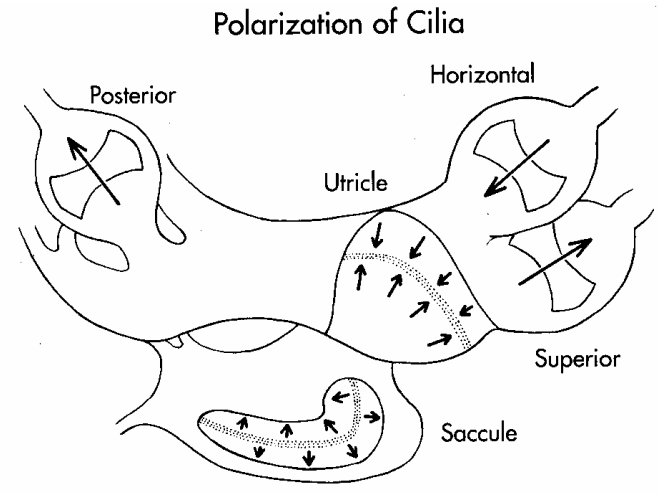

Figure 2: The direction of flow of endolymph in the labyrinth which stimulate the vestibular nerve

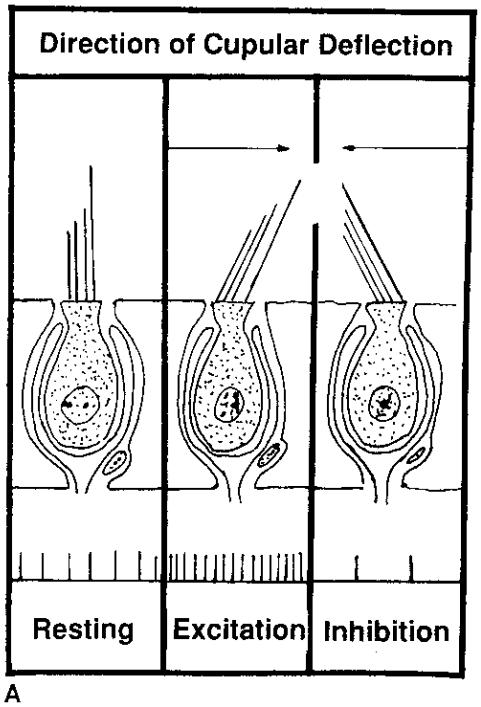

Figure 3: Direction of cupular deflection resulting in activity down the nerve fibre

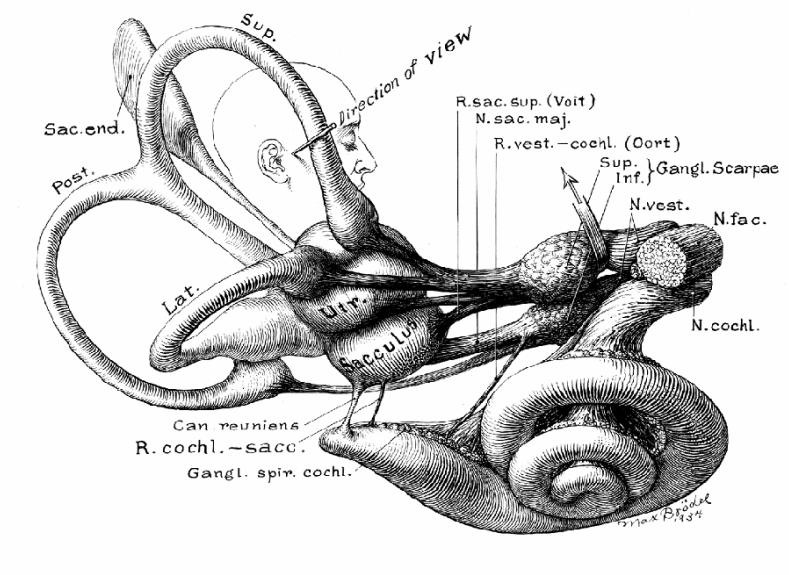

Figure 4: The vestibular organ

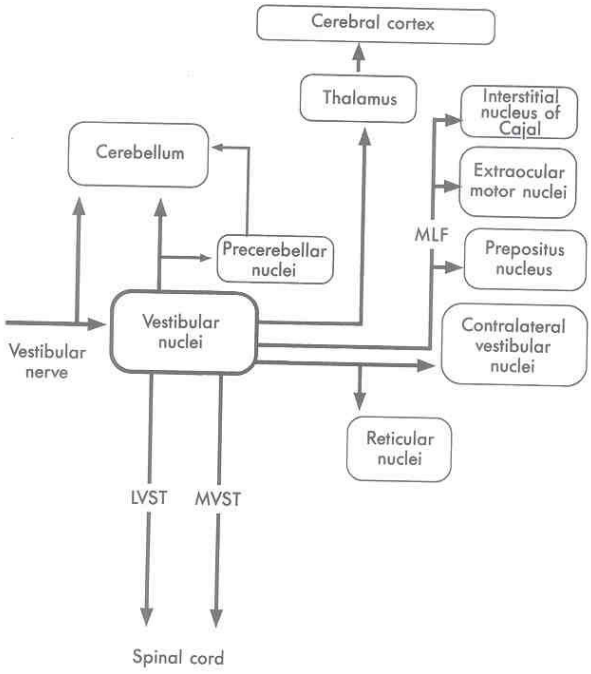

Figure 5: Central vestibular connections

The lateral semicircular canal is the most commonly tested because of its proximity to the middle ear. The lateral semicircular canal is excitatory to the contra lateral rectus and ipsilateral medial rectus and inhibitory to the contra lateral medial rectus and ipsilateral lateral rectus (Figure 6). The posterior semicircular canal is excitatory to the contra lateral inferior rectus and ipsilateral superior oblique and inhibitory to the contra lateral superior oblique and ipsilateral inferior oblique. The superior semicircular canal is excitatory to the contra lateral inferior oblique and ipsilateral superior rectus and inhibitory to the contra lateral superior oblique and ipsilateral inferior rectus. This is how the vestibulo ocular reflex is served.

INNER EAR FLUIDS

Endolymph: Among the extra cellular fluids endolymph has a unique ionic combination as it has a low sodium 5-25m mol and high potassium 150-160m mol causing it to resemble an intracellular fluid (Annico 1986, Smith 1954).

Perilymph: The site of production of perilymph may be as ultra filtrate of blood or CSF or both also known as Dual theory of origin (Kellerhals B 1979). CSF can reach the vestibule by means of the aqueduct or perivascular or perineural channels.

VESTIBULAR OCULAR REFLEX

The vestibulo-ocular reflex (VOR) represents one mechanism by means of which humans stabilize gaze, the clinical measurement of the oculomotor response to precise vestibular stimuli mediated through the semicircular canal-ocular reflex, enables quantification of labyrinthine function to be made. The principle function of the VOR is the control of eye position during transient head movement to maintain a stable visual image. In addition several other neural pathway that are independent of head movement contribute to eye movement

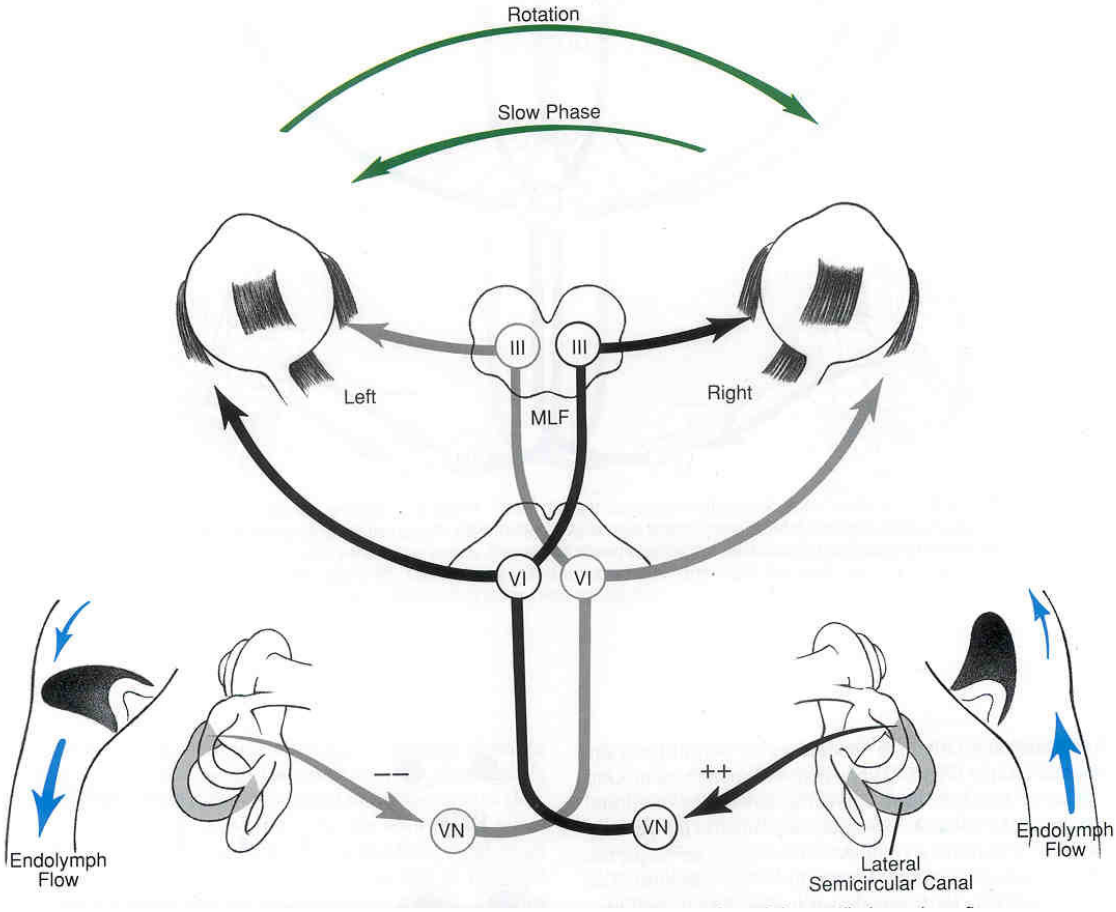

Figure 6: Lateral semicircular canal

control. The smooth pursuit system permits tracking of a visual target with a smooth continuous movement of the eye thereby providing a stable image projection to the fovea of the retina, the most sensitive area therefore greater clarity of image. The vestibulo-cerebellum (flocculus, nodulus, and posterior vermis) plays a dominant role in smooth pursuit (Carey, Santiana 2003)

The saccadic system of eye movement control provides the fast component during the production of jerk nystagmus. The function of the saccadic system is to reposition a visual target of interest onto the fovea with a single rapid eye motion(Leigh et al 1999). When a target of interest is moving outside the operating parameters of the smooth pursuit system, the saccade system facilitates the tracking ability by superimposing jerk movements onto the smooth movements. The difference between the position of the target on the retina and the desired position on the fovea is known as retinal slip (Neile et al 2000)

The optokinetic response is a combination of smooth pursuit and saccade mechanisms; the main purpose of this system is to provide clear visual images during sustained head movements. One system of oculomotar control is visual fixation. This is the active process of maintaining a fixed gaze on a target of interest. Although this system shares neurologic substrate with the smooth pursuit system, evidence suggests that it is a separate control system (Leigh 1999).

Vestibular adaptation after unilateral or bilateral vestibular loss takes place by central compensation. In cases of unilateral loss this can be by other sensory inputs like cervico-ocular reflex potentiation, alternate motor responses e.g. saccades for slow responses and strategies based on prediction and anticipation. Complete loss of vestibular function can be determined only by rotation testing, as caloric responses which simulate low frequency rotation stimulus may be absent in partial loss of vestibular function. This helps therapists to decide if patient needs a rehabilitation program for a deficient vestibular system or one that emphasizes sensory substitution (Zee 2000).

History and neuro-otological examination form the cornerstone of evaluation of a patient with giddiness. An appropriate history will help in making the all-important differentiation of a peripheral cause as opposed to a central cause. History also will differentiate from other systemic causes of instability like cardiovascular and metabolic. A good and comprehensive history must include the following (Hullar 2005).

- Does the patient have vertigo which is defined as hallucination of movement and indicates a lesion in the vestibular system
- Are symptoms episodic or continuous most vestibulopathies cause episodic symptoms
- Do symptoms indicate involvement of semicircular canals or otoliths sudden sensation of tilt and drop attacks are due to otolith dysfunction
- Thyroid disease diabetes anaemia autoimmunity hypo perfusion of brain, medications and arrhythmias all cause dizziness and vertigo
- Psychogenic causes like hyperventilation can cause episodic vertigo like symptoms
- Precipitating causes like position in BPPV sound in tullio phenomenon certain foods in migraine
- Exacerbation by head movements e.g. osscilopsia due to head movements indicate vestibular hypo function or vascular compromise of 8^{th} nerve complex

Associated symptoms like fluctuating hearing loss, tinnitus aural fullness as in Meniere's disease; slurring of speech, diplopia, and paresthesia in vertebra basilar insufficiency; aura in migraine and sweating and palpitations in panic attacks

EXAMINATION OF BALANCE SYSTEM :

Examination of patients with dizziness includes examination of the ear including otoscopy, tuning fork tests, examination of nose, nasopharynx, general physical examination, cranial nerves, and vestibulospinal and vestibulo-occular systems.

VESTIBULOSPINAL

Static imbalance

Romberg's: Patient stands with his feet together, eyes opened and eyes closed. If the patient sways it indicates proprioceptive loss or acute unilateral Labyrinthine dysfunction (Baloh 1995).

Walking: Patient is asked to walk on a straight line first with eyes open and then with eyes closed. Falls when tandem walking with hands stretched and eyes closed indicate ipsilateral horizontal canal dysfunction (Baloh 1995).

Unterbergers test – Patient is asked to stand in one place with hands out stretched and to march on the spot with eyes closed. If the patient moves more than 70 degrees to 100 degrees to one side, it indicates a paretic lesion on that side (Fitzgerald 1997).

Dynamic vestibulo-spinal function

Observation of postural instability during rapid turns or in response to external protuberance imposed by the examiner may indicate a dynamic vestibulo-spinal dysfunction. Examination of gait strength, reflexes, sensation in the legs and cerebellar function further assess the dynamic and cerebello-spinal function of the patient.

2.VESTIBULOOCCULAR SYSTEM

Nystagmus

These are involuntary rhythmic repetitive movements of eyeball.

Mechanism - Spontaneous nystagmus results from an imbalance of tonic signals arriving at the occulomotor neurons. Because vestibular system is the main source of occulomotor tonus, it is the driving force of most types of spontaneous nystagmus. The site of lesion may be in the peripheral vestibular pathway (labyrinths and vestibular nerve till root-entry zone)
or in the central vestibular pathways

The pathology may be located either in the peripheral vestibular system (sensory cell to the vestibular nuclei in the brainstem or central vestibular pathways

Lesions of peripheral vestibular system (labyrinth & 8th nerve) typically interrupt tonic afferent signals originating from all of the receptors of one labyrinth so that the resulting nystagmus has combined torsional horizontal and vertical components. The horizontal component dominates because the tonic activity from the intact vertical canals and otoliths partially cancel out. Gaze in the direction of the fast component increases the frequency and amplitude. Gaze in the opposite direction has the reverse effect.(Alexander's Law). The slow phase is linear resulting in a saw-toothed wave-form. Peripheral vestibular nystagmus (PVN) is strongly inhibited by fixation. Unless seen within a few days of the acute episode, spontaneous nystagmus will not be present when fixation is permitted.

Central type of nystagmus

These are due to dysfunction of central vestibular pathways have been classified in various ways

***Congenital spontaneous nystagmus*:**

This is a purely horizontal nystagmus which disappears / markedly decreases with loss of fixation as well as during convergence.

Periodic Alternating nystagmus

This nystagmus changes direction at regular intervals (1 to 6 minutes) with null periods (where nystagmus is minimal or absent) between each half cycle varying between 2 to 20 secs. It may be found in with various conditions such as encephalitis, brainstem ischemia, syringobulbia, syphilis, trauma and as a congenital disorder

Inspection of spontaneous nystagmus:

The eyes are observed with the and without Frenzel's lens when the eyes are looking straight. Frenzel's Lens (+20 dioptre lens) is used to differentiate spontaneous nystagmus of peripheral origin (enhances without optic fixation) from central vestibular nystagmus which does not enhance on removal of optic fixation.

Spontaneous nystagmus. The difference between peripheral and central nystagmus is shown in Table 1

Table:1 DIFFERENTIATION BETWEEN SPONTANEOUS NYSTAGMUS OF PERIPHERAL AND CENTRAL ORIGIN (Baloh et al 1990)

	peripheral	Central
appearance	Combined/torsional/horizontal	Often pure-horizontal/vertical/ torsional
fixation	Inhibited	Usually little effect
gaze	Unidirectional(Alexander's law)	May change direction
mechanism	Asymmetric loss of peripheral vestibular tone	Imbalance in the central occulo-motor tone; may be OKN /pursuit*
location	Labyrinthine or vestibular nerve	CNS/brainstem/ cerebellum

Gaze-holding nystagmus (Baloh 1990).

Patient is asked to maintain eccentric gaze for 30 sec from central orientation (Drift up to 15-sec normal). Presence of gaze-holding nystagmus is a hallmark of cerebellar floccular and medial vestibular lesion.

In cerebello-pontine angle lesion large amplitude gaze-evoked nystagmus is seen. Drugs like, hypnotics, sedatives, anxiolytics will show low amplitude gaze-evoked nystagmus.

The alternate cover test / Madrox rod (Baloh 1990).

Eyes of the patient are alternatively covered with a card looking for a vertical or horizontal corrective movements as an index of misalignment.

Saccades:

Patient is asked to alternatively fixate (with head still) on the examiner's nose and finger, which is moved to different locations 15^0 from primary position. Parameters like velocity, accuracy, and initiation time are looked for.

Smooth pursuit

Patient is asked to follow a slowly moving target no faster than 20^0 per sec. Asymmetries in horizontal tracking as represented by the presence of more corrective saccades in one direction than other is looked for.

Head shaking nystagmus (Sawovaros 1999)

With Frenzel's lens in place, patient is asked to shake head about 30 times horizontally with chin placed about 30 degrees downwards. On stopping the head shaking abruptly presence of any nystagmus is looked for. This test checks dynamic vestibular function

In unilateral vestibular lesion a vigorous nystagmus with slow phase initially directed towards the side of lesion and then a "cross-coupled nystagmus" may be noticed. A mechanical disturbance in the labyrinth e.g. debris adherent to the semicircular canal/abnormality of cupula itself can cause head-shaking nystagmus. It is an objective sign of an imbalance somewhere in the vestibular system

Head thrust test (Halmagvi1988)

Patient is asked to look carefully on examiners nose. A brief high acceleration horizontal head thrust is done.

In unilateral vestibular failure a slow phase of abnormally low amplitude nystagmus will be evoked in response to head thrust towards a point of fixation.

Positioning testing (Alford 1972)

***Dix – Hallpike manoeuvre* (figure 7)**

Patient sits upright on examination table and turns chin 45 degrees towards right shoulder. Patient is brought straight back rapidly into a right head hanging position. This position is maintained for at least for 30 sec.

Table 2 shows the differences between benign paroxysmal positional vertigo and positional vertigo of central origin

Table:2 COMPARISON OF THE POSITIONAL NYSTAGMUS OF BENIGN PAROXYSMAL POSITIONAL VERTIGO (BPPV) WITH THAT OF CERTAIN LESIONS OF THE CENTRAL NERVOUS SYSTEM (Baloh 1990 and AG Kerr et al 1997)

	BPPV	**CNS**
Latent period	A few seconds	Nil
Distress	Severe vertigo	Nil
Direction of nystagmus	Rotatory with the fast component towards the under most ear	Variable
Duration of nystagmus	Less than 30 sec	Persists while position maintained
Fatigability	Nystagmus and vertigo stops with repeat testing	Nystagmus persist with repeat testing
Mechanism	Otolith debris moving in semicircular canal	Damage to central ocular pathways
Localization	Posterior or horizontal semicircular canal	Brainstem or cerebellum

Valsalva induced nystagmus (Baloh 1990)

The patient is asked to do Valsalva's manoeuvre with Frenzel's lens in position and nystagmus is looked for. It is seen in cranio-cervical junction anomalies, perilymphatic fistulas, other abnormalities of ossicles, oval window, semicircular canals and otoliths.

Hyperventilation

The patient is asked to breathe deeply for about 30 to 60 seconds, after which the eyes are observed under Frenzel's lenses. Patients with chronic anxiety describe sensation of light-headedness, faintness, giddiness, oral numbness paraesthesias of the extremities, lump in throat, and tightness in chest (Drachmann et al 1972). Hyperventilation causes presyncopal light-headedness by lowering the carbon dioxide content of the blood, thus producing constriction of the cerebral vasculature (Gotch et al 1965) Patients with demylinating lesions

Figure 7: Dix – Halpikes Test

of vestibular nerve e.g. acoustic tumour, compression by a small blood vessel, multiple sclerosis may show hyperventilation induced nystagmus (Leigh et al 1999). The metabolic consequence of hyperventilation (alkalosis, changes in ionized calcium) may provoke nystagmus either by alternating conduction in peripheral (e.g. vestibular schwannoma) or central (e.g. multiple sclerosis) vestibular pathways, or by altering central compensation. For example, a vestibular schwannoma may cause focal demyelination of the vestibular nerve, reducing conduction across this segment. Central mechanism compensate for the resulting vestibular imbalance (minimal or absent spontaneous nystagmus). Hyperventilation, however, improves conduction, increasing input from the lesioned side and making the central compensation no longer appropriate. The result is a nystagmus with slow phase directed away from the side of the lesion (Walkerand and zee 2000)

Fistula test

This test is done by tragal compression or insufflation using Siegel's speculum. Nystagmus is seen in otitic syphilis, perilymph fistula, and dehiscence of the semicircular canals caused by cholesteatoma or congenital anomalies.

CLINICAL EVALUATION OF HEARING :

Hearing disorders are classified as conductive, sensorineural, and central based on the anatomic site of lesion. (Beagley 1981silverman 1978)

CONDUCTIVE HEARING LOSS:

The tympanic membrane and ossicles act as a transformer amplifying airborne sound and efficiently, transferring it to the inner ear fluid. If this normal pathway is obstructed, transmission may occur across the skin and through the bones of the skull, but at the cost of considerable energy loss.

The causes of conductive hearing loss are impacted wax, otitis media (suppurative otitis or serous otitis), otosclerosis, perforations of tympanic membrane, trauma, congenital malformation of the external and middle ears, and tumours of the temporal bone.

SENORINEURAL HEARING LOSS:

Sensory neural hearing loss results from lesions of the cochlea and/or the auditory division of the eighth cranial nerve. The cochlea analyze the frequency content of the sound, the high frequency sounds stimulate the sensory cells of the basal turn whereas for low frequency sounds maximum stimulation occurs at the apex. The common cause of acute unilateral hearing loss is infection of inner ear (labyrinthitis). It can either be viral (measles, mumps, infectious mononucleosis) or bacterial. The other causes are trauma and vascular occlusive disease. Relapsing unilateral sensorineural hearing loss associated with tinnitus, fullness in ear, and vertigo is typical of Meniere's disease. Ototoxic drugs produce a subacute hearing loss. Acoustic neuromas characteristically produce a slowly progressive unilateral sensory neural hearing loss. The progressive bilateral hearing loss associated with advanced age is called as presbyacusis.

CENTRAL HEARING DISORDER:

Central hearing disorders results from lesions of the central auditory pathways: the cochlear and dorsal olivary nuclear complexes, inferior colliculi, medial geniculate bodies, auditory cortex in the temporal lobes and their interconnecting afferent and efferent fibre tracts. Lesions involving the nerve root entry zone or cochlear nucleus can result in unilateral hearing loss for pure tones. Because approximately 50% of afferent nerve fibres cross central to the cochlear nucleus, this is the most central structure in which a lesion can result in unilateral hearing loss.

AUDIOMETRIC TESTS:

The audiologist is an integral member of the team, evaluating patients with dizziness. The audiologists role in to do a comprehensive audiologic evaluation which includes pure tone testing, speech audiometry, and immittance testing. The audiogram is performed in a sound proof room with calibrated audiometric equipment

Air conduction testing (AC): A series of frequency specific pure tones (250 – 8000 Hz) is presented via ear phones, asking the patient to respond each time he hears the stimulus. The audiologist finds and maps the threshold, defined as the softest intensity level at which a patient can hear the tone at least 50% of the time. The ears are tested individually, and the threshold is obtained in dB hl at 250,500, 1000, 2000, 4000 and 8000 Hz.

Bone conduction testing (BC): The second part of comprehensive audiologic evaluation is testing bone conduction. This involves placement of a bone conduction vibrator on each mastoid individually and finding thresholds at various frequencies. Bone conduction testing differs from air conduction in its mode of presentation; in addition it by passes the outer and middle ear, delivering the stimuli directly to the cochlea of the inner ear. In certain cases (e.g. with conductive loss and asymmetry between ears), the stimulus (tonal / speech) in the test ear can travel through the skull and around the head to be perceived by the non-test ear. To obtain valid test results in the test ear, the audiologist may find it necessary to present a noise to the non-test ear, preventing it from participating in the test. This is called masking (Valente 2001)

***Caloric test*:**

Marie Jean Pierre Flourens in early 1800 provided the first scientific clues that semicircular canals were involved in balance. In experiments on the pigeon he showed that a lesion in the horizontal semicircular canal caused the animal to turn in the vertical axis while a lesion in the posterior canal caused the animal to

roll over. Flourens further observed that the hearing was preserved in spite of lesion on the semicircular canals and that direction of movement was same as that of the canal divided. (Lustig 2000)

Nobel prize winning scientist Robert Barany introduced into clinical examination caloric testing and also explained different types of nystagmus and their central causes.

In 1870 Goltz concluded that the semicircular canals were responsible for balance only. Barany in 1906 used 10 to 20 ml of ice water to cause nystagmus. This caused past-pointing and drift (walking towards irrigated side) also along with autonomic symptoms of nausea and vomiting (Nelson 1969).

Kobrak in 1920 in order to minimize patient discomfort used smaller volumes of water at temperatures closer to body temperature. He used 5 to 10ml of water at 27 degrees and if no response was found lowered the temperature stepwise to 20 degrees. This required a temperature controlled water source was time consuming but reduced patient discomfort significantly

Hallpike and Fitzgerald in 1942 (Nelson 1969) introduced the accepted Bithermal Caloric test in which the ears were irrigated using water for forty seconds using temperatures 7 degrees above and below body temperatures. This test caused only moderate discomfort was very sensitive and specific and evaluates both central and peripheral vestibular system.

Linthicum in 1964 described a method using only 2 cc of ice water .He progressively doubled the amount of ice water if no response was found. Thus he attempted to find the threshold amount of ice water required to induce nystagmus.

Caloric test is useful in determining the responsiveness of the labyrinth and is one of the few tests that allow the single labyrinth to be tested independently. Caloric test relies on alternatively heating and cooling the labyrinth by using water or air. Being closest to the plain of the temperature gradient it is the horizontal canals which is tested.

Caloric stimulus of the labyrinth cause a response in two ways. The first is a convective component with a temperature gradient causing a density difference. When the horizontal canal is oriented in plane of gravity either by raising 30 degrees or lowering 60 degrees from the upright position the gravity allows denser fluid to go lower and less denser to go higher this movement deflects cupula and results in nystagmus. A nonconvective component has been proved by demonstration of caloric effect in space where there is microgravity environment and no convective effect. This may be due direct caloric effect on hair cells or cupular displacement due to pressure changes in membranous duct. (John Stahle 1990)

Water at 30 and 44 degrees are used and irrigation is for 60 seconds cerumen to be removed water to be avoided in perforation of tympanic membrane mental arithmetic is done for concentration optic fixation is removed with frenzels glasses (Luxon 1997)

In a study (Nelson 1969) the minimal ice-cold caloric test (MIWCT) with established vestibular caloric test procedures, Video oculographic investigation was performed in 22 healthy subjects using ice water (.5, 1 and 2ml) classical caloric test procedures (CCTP) and cold air at 27 degrees. MIWIT was associated with significantly later onset of nystagmus and significant prolongation of nystagmus. In contrast to air at 27 degrees, a significant Spearmans correlation was found between MICWT and established CCTP in respect of essential nystagmus parameters like frequency amplitude and SPV. Further MICWT showed a higher sensitivity and specificity with regard to detection of

canal paresis based on Jongkees formula compared to air at 27 degrees. This study showed that the minimal ice-cold irrigation study could be used safely as a screening test

In another study (Becker GD 1979) to assess the screening value of monothermal caloric tests the following observations were made

A valid screening caloric test must decrease examination time increase patient comfort and maintain high degree of sensitivity in predicting Bithermal caloric results.

Comparing monothermal warm and cold irrigation false negative results were obtained in 14 and 25 % of irrigations and false positive irrigations in 22 and 15 % of irrigations. This lead to some patients not getting Bithermal tests and others getting unnecessary Bithermal tests.

This lack of sensitivity limits the use of monothermal tests as screening tests.

In a study (Nelson 1969) Linthicum method was explored further in normal and vertiginous patients by not only measuring the amount of ice water needed but also the duration and intensity of response. This was to evaluate the overall stimulus strength and to assess the usefulness of the method as a screening test.

Three fourths of the normals demonstrated nystagmus with .2 cc ice-cold water confirming the conclusion of Linthicum. The response duration between the two ears was relatively constant within 10 to 15 seconds of each other. The response duration was compared to standard Hallpike 30 degree stimulus for 30 seconds. The mean response after .2 cc was 99.3+/- 22.5 for right ear and 99.5+/- 20.2 for left ear. After 30-degree stimulation it was 131.7+/-16.3 for the

right ear and 130.8 +/-14.7 for the left. This 30 second duration difference was found to be statistically significant by student T test (p=<0.001). Even in vertiginous patients stimulus with .2 cc evoked lesser response than 30 degree 30 second stimulation . Thus .2 cc stimulus is a weaker stimulus by duration criterion than 30 degree stimulus.

Since the strength of stimulus is more represented by the slow phase velocity, the measurement of slow phase velocity showed it to be 12.7 for 30 degree and 3.2 for .2 cc stimulation. But the discomfort to the patient in terms of subjective vertigo was less with.2cc than 30-degree stimulus. A further conclusion made was when a marked asymmetry was noticed in the two ears with .2 cc stimulus the phenomenon of directional preponderance should be considered and when different amounts of water was needed for a response for example .2cc on right and .8cc on left then the phenomenon of canal paresis should be thought of. In this study the minimal ice-cold irrigation was found to need >.4 cc in cases of acoustic neuroma in 85% of proven cases.

Hence it was concluded that minimal ice cold irrigation test is a useful screening test.

Electronystagmography

This test has been used for clinical testing for nearly forty years. The test consists of a battery of tests collectively known as Electronystagmometry (Sheperd 2000).It is one of the standard investigations used in the assessment of patients suffering from vertigo and equilibrium disorders.

Principle of ENG

The retina is charged negatively as against the cornea. Therefore there is a corneo-retinal potential (Figure 8) present the electrical axis of which coincides with optical axis of the eye.

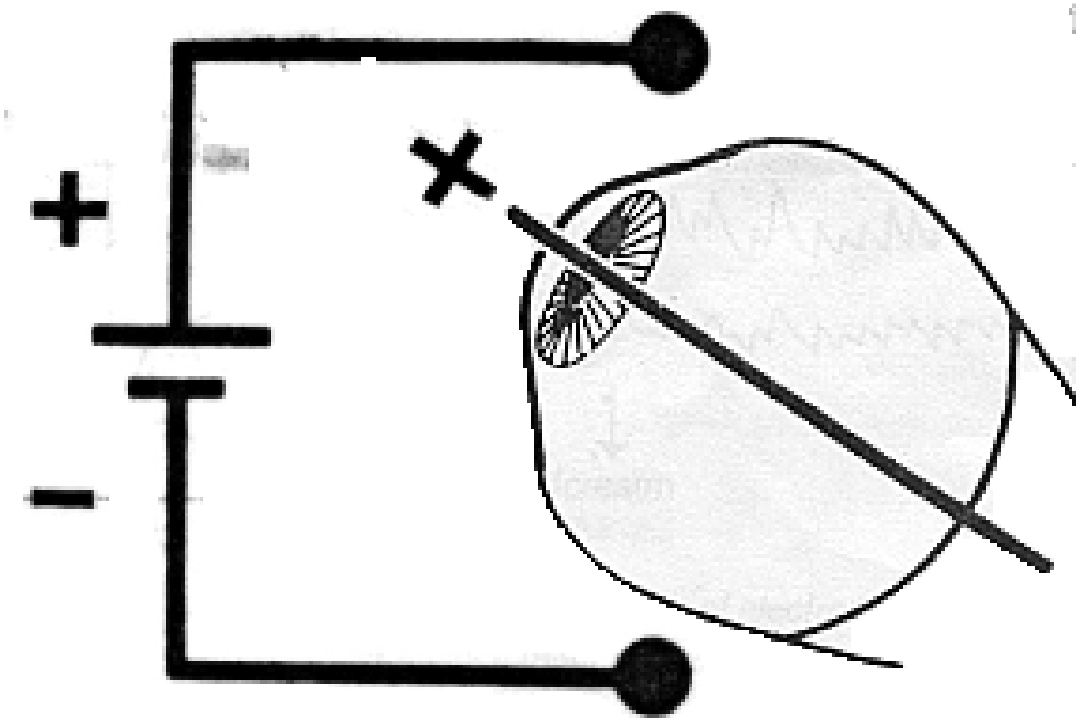

Figure 8: Corneo-retinal potential

This potential is in microvolt range. A movement of eyeball causes a movement of the electrical field and the currents so created can be picked up by electrodes applied to skin around the eyeballs. The voltage measured is proportional to the amplitude of the eye movement.

The potentials are greatly amplified and recorded directly on a running strip of paper similar to ECG (usually a AC amplifier is used)

By convention, movements of the eye to right and up are recorded as an upward deflection, left and down as downward deflection.

The ENG Machine

It can be single channel - records conjugate horizontal eye movement
Double channel - can record horizontal movement of each eye separately
Or a multichannel - which can record both horizontal and vertical eye movements

The more recent machines have been computerized so that all parameters are computed and calculations available immediately.
The most recent machines make use of an infrared camera, which picks up the nystagmus and records into a computer and parameters calculated and made available instantly.

Test conditions

Patient from 5 yrs or older can undergo this test. It is important to ensure that the patient is alert (not been on labyrinthine sedatives/hypnotics/tranquilizers or alcohol for at least 48 hrs prior to the test). It is important to tell patient not to apply any powder or creams to the face prior to the test.

Patient undergoes a full ENT and examination prior to the test to exclude any ear wax/perforations/mastoid cavity/infections etc

Test should be done in a room with no electrical disturbances/interference and must have an even lighting with ability to darken surrounding.

Patient should be seated in a comfortable positioning chair for head to be positioned at 30 degrees above horizontal

Preparation of patient

Skin is cleaned well with spirit and electrode jelly applied and electrodes fitted on both the outer canthi of the eyes with the neutral electrode on the fore head.

A soft rubber catheter is applied to each deep meatal wall under vision

Calibration is done in two ways electrical Bio-calibration (pendular test)

Electrical calibration

An impulse of known voltage is fed in and machine so adjusted that a 200 microvolt signal produces 10 mm deflection of the recording needle. (Ambient electrical disturbances can produce movements of recording needle but these usually have amplitude of less than 20 micro volts. Therefore any beat less than one mm should not be considered as nystagmus). This is already done by the manufactures.

Bio calibration done by using a calibration bar

- Done in order to interpret ENG
- A standard angle of eye deviation is represented by known amplitude of pen deflection.
- patient is asked to follow movement of a pendulum moving through an angle of 40 degrees of eye movement (20 deg to right, 20 deg to left) in front of the patient.
  I.e.: xy =20 mm=40 degrees

Therefore 1mm of pen deflection is 2 degrees of eye movement

This test called pendular test can be used to calculate amplitude and slow phase velocity of nystagmus

It also gives information about proper fixing of electrodes and correctness of polarity of fixed electrodes

ENG test battery

Spontaneous nystagmus

Definition - Nystagmus present with head upright and eyes centered.

Technique - The patient is seated upright and eyes cantered. Recording is taken with eyes open for one minute and closed for one minute.(Latter 30 secs of each minute taken for calculation)

Gaze- evoked nystagmus

Nystagmus which appears on gaze deviation in one or more directions and is not present in imposition

Technique

Patient is asked to look 30 degrees to right and to left, up and down. Recording of eye movements is done in each gaze position for one minute.

- Pathological if amplitude >4 degrees

Types of nystagmus

Symmetric - equal amplitude to right and left

- Commonly produced by ingestion of drugs e.g. phenobarbital, phenytoin, alcohol and diazepam

- also seen in pts with myasthenia gravis, multiple sclerosis and cerebellar atrophy

Asymmetric - always indicates structural brain lesion
- In Brun's nystagmus (found in large c-p angle lesion which causes compression of brainstem) the larger amplitude nystagmus is usually directed towards side of the lesion

Rebound - nystagmus that disappears or reverses direction as the lateral gaze position is held
- occurs in patients with cerebellar atrophy, and focal structural lesions of cerebellum (only variety of nystagmus thought to be specific for cerebellar involvement)

Dissociated-results in lesions of medial longitudinal fasciculus(MLF). On adducting eye lags behind and develops a low amplitude nystagmus while abducting eye overshoots the target and develops a large amplitude nystagmus. E.g. demyelinating diseases, unilateral -usually vascular diseases of brainstem

Caloric testing (Baloh 1990)

Patient is laid supine, with head end elevated by 30 degrees from the horizontal position.20 ml of water at 44 degrees and 30 degrees are used for irrigation in the following order. Right 44 left 44 right 30 left 30.
An interval of 8 minutes is given between each successive irrigation.
Patient is kept alert during procedure using simple arithmetic (eg:subtracting 7 from 700).

Parameters calculated

1. ***Duration of nystagmus*** was not found to be very satisfactory because

a) The induced nystagmus does not stop very abruptly and endpoint is difficult to determine

2. *Maximum slow phase velocity*- true representative of vestibular activity
There is a large intersubject variability. Therefore the intrasubject measurement is more useful clinically

The formula to calculate vestibular paresis

$$\frac{(R30+R44)-(L30+L44)}{R30+R44+L30+L44} \times 100 \;\; \% \text{ Vestibular Paresis (VP)}$$

For directional Preponderance the formula is

$$\frac{(R30+L44)-(R44+L30)}{(R30+L44+R44+L30)} \times 100 \;\; \% \text{ Directional Preponderance (DP)}$$

Table 3 shows the probable site of lesions in Bithermal test results. A canal paresis can indicate reduced response of the semicircular canal. A directional preponderance in presence of a spontaneous nystagmus can be due to an uncompensated or overcompensated vestibular function. In absence of spontaneous nystagmus, it may be indicative of a central lesion.

Table 3: Interpreting the result of Bithermal caloric testing

	location of lesion	mechanism
vestibular paresis	labyrinth, 8th nerve	decreased peripheral sensitivity
directional preponderance	non localizing	tonic bias in vestibular system
hyperactive responses	cerebellum	loss of inhibitory influence on vestibular nuclei
dysrhythmia (marked beat to beat variability in caloric induced nystagmus)	cerebellum	loss of inhibitory influence on pontine nuclei
impaired fixation suppression	CNS pursuit pathways	interruption of visual signals on way to oculomotor neurons
perverted nystagmus (vertical/oblique nystagmus produced by stimulation of horizontal SCC)	4th ventricular region	disruption of vestibular commissural fibres

Limitations include that it does not test full dynamic range of the vestibular system. Measures the function of the lateral semicircular canal only, and it is considered to be a very low frequency test for vestibular function.

Vestibular evoked myogenic potential are considered a test of the otolith as this response is shown to arise from the saccule. A burst of activity in the ipsilateral sternomastoid has been recorded using a electromyography to sound at 95 db spl.

Computerized dynamic posturography the standard test battery includes postural responses to platform movements the motor control test and sensory organization test due to results of manipulation of visual or somatosensory information there are six test conditions in the sensory organization test.

The computerized posturography is useful in guiding a rehabilitation program and in organizing a relevant exercise program. It has been used to determine the need for placement of ventriculoperitoneal shunts in patients with high CSF pressures causing instability. This test provides documentation for postural responses for malingering and exaggeration of disability and conversion disorders. (Biswas 2006)

MATERIALS AND METHODS

This is a descriptive, cross-sectional study of normal volunteers (controls) and patients with rotatory vertigo (symptomatic patients) as the study arm to determine the usefulness of cold caloric test as a screening test for patients with vertigo.

A sample size of 30 normal and 30 cases with vertigo will be sufficient to provide results for a test with a Sensitivity of 87% (95% CI: 73- 95) and a Specificity of 50% (95% CI: 27 - 72), calculated for a type I error of 5% and a power of 80%.

The normals (controls) were volunteers selected from the staff and students of Christian Medical College. A standard questionaire was administered to them to ensure they were eligible to be included (Annexure 1). They were also asked to consent for undergoing tests and given an information sheet (Annexure 2), prior to the test.

Inclusion and exclusion criteria for selecting the controls and symptomatic patients are shown below.

Inclusion criteria:

For Normals: Persons 18 yrs to 65 years, with no history of ear-related complaints (hearing loss, tinnitus, giddiness,) with normal hearing on pure tone audiogram.

For symptomatic patients: All patients attending the audiovestibular clinic with a complaint of giddiness.

Exclusion Criteria:

For Normals

History of medication with potentially ototoxic drugs, exposure to excessive noise, history of ear discharge/ head trauma, systemic illnesses like diabetes, hypertension, hypothyroidism.

For symptomatic patients

Tympanic Membrane with central perforation, active otitis externa and patients on labyrinthine sedatives less than 48 hrs before the tests.

All controls and patients underwent a detailed history and otoneurological examination including examination of ears to rule out presence of wax or tympanic perforation. Their hearing status was established with pure tone audiometry and impedance audiometry done in a sound proof room.

Those who fitted into the inclusion and exclusion criteria underwent the minimal cold irrigation test (Baloh et al) and Bithermal caloric test as described by Fitzgerald and Hallpike (Nelson 1969).

Minimal cold irrigation test.

The test was done by a third person, not involved in recruiting or interpreting results. The patient was made to lie down supine, with the head flexed at an angle of 30 degrees to the horizontal, so as to place the horizontal semi circular canal in a vertical position. Under direct visualization of the ear drum, two cc of ice water (obtained by adding ice cubes to tap water until it reached 4 deg C) was infused into the ear canal against the tympanic membrane through a small rubber hose. The ear being infused was tilted up for 15 seconds after infusion, to be certain that the water stayed against the drum and then the head brought back to its original position. The patient was asked to count backwards from 100 in twos, to keep the subject alert. Nystagmus was

observed through Frenzel's lens (+20 diopter lens) and its duration measured using a stop watch. Moderate lighting was used to prevent optic fixation .

Bithermal caloric test

The patient was made to lie down supine with head flexed at a 30 degree angle to the horizontal so as to place the horizontal semi circular canal in a vertical position. Each ear was infused with water at 30 deg C and 44 deg C for 30 seconds, using a soft red rubber catheter fixed in such a way that the irrigation would impinge on the tympanic membrane. The patient was asked to count backward in twos, from 300 (to keep the patient alert). The duration of nystagmus was recorded by means of an infrared camera of the video nystagmography machine. The same was repeated after 8 minutes in the opposite ear. Both the ears were sequentially irrigated with warm water at 44 deg C, with an interval of 8 minutes by a third person who was not involved in recruiting or interpreting results. The 44 deg C irrigation was done in order to calculate canal paresis and directional preponderance using Jongkees formula.

$$\frac{(R30+R44)-(L30+L44)}{(R30+R44+L30+L44)} \times 100 = \% \text{ Vestibular Paresis (VP)}$$

$$\frac{(R30+L44)-(L30+R44)}{(R30+R44+L30+L44)} \times 100 = \% \text{ Directional Preponderance (DP)}$$

Recruitment was stopped as soon as the sample size was reached in each group. Data was entered on an excel spreadsheet and analysed using SPSS v 12 .The normal values for cold caloric test was taken as +/- 2 standard deviations (SD) of the mean values of the 30 normal volunteers.

Similarly, the normal values for Bithermal caloric tests were also calculated by taking +/- 2 SD from the mean.

Frequencies and cross tabulations were done and Chi square where appropriate. Sensitivity, specificity and predicative values of the cold calorie test were calculated.

RESULTS

There were 30 normal volunteers and 30 symptomatic patients; 15 (50%) normals were females and 15 (50%) were males while in the symptomatic patients, 12 (40%) were females and 18 (60%) were males.

Figure 9 shows their age distribution. Most volunteers were in the age group 25 to 34 years while symptomatic patients were more in the older age groups.

Table 4 shows the age and sex distribution in the study groups. Age groups 25 to 34 and 55 or more had the largest numbers. Only a small proportion (6.7%) were younger than 25. There were more males than females in this study.

Table 4. Age and sex distribution of normal and symptomatic groups

Age Group	Females (%)	Males (%)	Total
15 to 24	3 (11.1)	1 (3.0)	4 (6.7)
25 to 34	9 (33.3)	8 (24.2	17 (28.3)
35 to 44	8 (29.6)	5 (15.2)	13 (21.7)
46 to 54	3 (11.1)	7 (21.2)	10 (16.7)
55 and above	4 (14.8)	12 (36.4)	16 (26.7)
Total	27	33	60

Table 5 shows the mean duration of nystagmus with cold caloric test among normals, 2 SD and mean ± 2 SD in the right and left ears.
The mean duration of nystagmus for both right and left was more than 1½ minutes.

Table 5. Cold caloric test results in normal volunteers.

Right ear	**Left ear**
Mean duration: 100.7	Mean duration: 103.2
2 SD: 54.75	2 SD: 49.0
Mean – 2 SD: 45.9	Mean – 2 SD: 54.2
Mean + 2SD: 155.4	Mean + 2SD: 152.2

Table 6 shows the results of Bithermal caloric tests among normal volunteers, standard deviation and mean +/- 2 standard deviations The mean +/- 2 SD for VP frequency was 21.6; for VP spv was 24.93; for DP frequency was 19.99 and for DP spv was 32.13

Table 6. Results of Bithermal caloric tests among normal volunteers

	VPfreq (%)	**VPspv (%)**	**DPfreq (%)**	**DPspv (%)**
Mean	8.21	9.69	7.44	12.85
Std. Deviation (SD)	6.53	7.62	6.27	9.64
2 SD	13.05	15.24	12.55	19.28
Mean + 2 SD	21.26	24.93	19.99	32.13

Table 7 shows the clinical diagnosis of patients presenting with giddiness. The largest group were neurological with migrainous vertigo being the commonest diagnosis. Metabolic abnormalities were found in 8 out of the 30 patients which may have contributed to the symptom. Endolymphatic hydrops was next commonest with 5 patients.

Table 7 - Clinical diagnosis of symptomatic patients

Clinical diagnosis	**number**
BPPV	3
Neurological (Migrainous vertigo / meningitis)	10
Endolymphatic Hydrops	5
Cardiovascular (postural hypotension / VBI)	
Metabolic abnormalities (DM/ dyslipidaemia/ hypothyroidism)	8
Ototoxicity	1
Cochlear Otosclerosis	1
AVE poor vestibular compensation	2

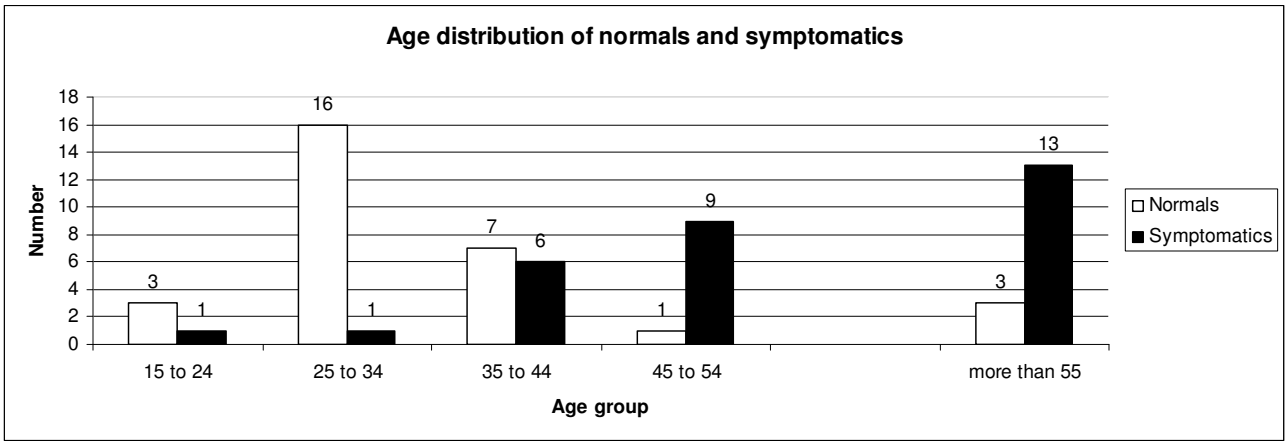

Figure9: Age distribution of normal volunteers and symptomatic patients

Table 8A. Symptomatics and of canal paresis (frequency)
(mean ± 2 SDSD of the normals)

Value (frequency)	Number	Percent
<21.27	25	83.3
>21.26	5	16.7
Total	30	100.0

Table 8B. Symptomatics and frequency of canal paresis
(mean ± 2 SD of the normals)

Value (SPV)	Number	Percent
<24.94	12	40.0
>24.93	18	60.0
Total	30	100.0

Tables 8A and 8B show the number of symptomatic patients with canal paresis based on frequency of nystagmus and slow phase velocity respectively (during cumulative period) using values generated from normals. Of the 23 patients with canal paresis, 18 were detected by SPV and 5 were detected by frequency parameter.

Tables 9A and 9B show number of symptomatic patients with directional preponderance based on frequency of nystagmus and slow phase velocity respectively (during cumulative period) using values generated from normals. Of the 14 symptomatic patients with directional preponderance 8 were detected by SPV and 6 were detected by frequency.

Table 9A. Symptomatics and frequency directional preponderance

Value (Frequency)	Number	Percent
<20.0	24	80
>19.99	6	20
Total	30	100

Table 9B. Frequency directional preponderance (SPV) among symptomatics.

Value (SPV)	Number	Percent
<32.14	22	73.3
>32.13	8	26.7
Total	30	100.0

Table 10 shows a comparison of results from cold caloric and Bithermal caloric tests among symptomatic patients. Sensitivity of the cold caloric test was 3/24= 12.5% and specificity was 5/6=83.3%

Table 10. A comparison of results from cold caloric and Bithermal caloric tests among symptomatic patients.

	Bithermal test positive	Bithermal test negative	Total
cold caloric test positive	3	1	4
cold caloric test negative	21	5	26
	24	6	30

Sensitivity of the cold caloric test was 3/24= 12.5% and specificity was 5/6=83.3%

Table 11 shows a comparison of results obtained from structured questionaire and the Bithermal caloric tests among 30 normals and 30 symptomatics.

Table 11. Comparison of results obtained from structured questionaire and the Bithermal caloric tests among normals and symptomatics.

	Bithermal test positive (%)	**Bithermal test negative (%)**	**Total**
Symptomatics (questionnaire) positive	24 (40)	6(10)	30
Normals (asymptomatic) (questionnaire)	3 (5%)	27(45%)	30
Total	27	33	60

DISCUSSION

This study was undertaken to evaluate the usefulness of a frequently used test i.e. cold irrigation test as a screening procedure for detecting patients with giddiness who may need further evaluation or to reassure the patient with giddiness that there was no abnormality with the vestibular system. The cold irrigation was evaluated against the available gold standard test which is the Bithermal caloric test.

Controls were used to establish the normal values in the population. As can be seen from the Figure 1, the control group age group was considerably younger than the symptomatics. This was because most controls were student volunteers. The symptomatics as expected were older, with risk factors that predispose a person to develop giddiness.
Male predominance in the study group may be attributed to the pattern of hospital visit to a tertiary care centre. The duration of nystagmus after cold irrigation in normal volunteers were very similar in both ears and may be related to the fact that symmetrical activity occurs in both the labyrinths in a normal person.

This study (Table 6) had VP frequency, VP spv, DP frequency, DP spv similar to observations in other centres (Biswas 2006)

The clinical diagnosis reflects the types of patients who come to the vertigo clinic in a tertiary clinic. Migrainous vertigo is one of the commonest causes of giddiness followed by metabolic abnormalities like diabetes mellitus and dyslipidaemia.

Bithermal caloric test interpretation is based on a formula by Halpike and Fritzgerald that eliminates the large inter-subject variability. Bithermal caloric test behaved the way it should have when applied on normals and symptomatics

since it showed that a significant proportion of symptomatics were identified as positive by this test.

In our study, we found that the sensitivity of cold caloric test was 12.5% and the specificity was 5/6=83.3%. Keith et al (1991) in their study to re-evaluate the monothermal caloric test by examining the correlations between unilateral weakness derived from Bithermal caloric stimulation compared to monothermal caloric results, found that while predicting normal or greater than 20% unilateral weakness, both warm and cool monothermal calorics have greater than 85% efficiency, with specificity greater than 94% and sensitivity greater than 64%. However, the false-negative rate is 29% for warm and 36% for cold caloric test. They concluded that the high rate of false-negative findings indicates that screening tests have no place in a diagnostic battery, especially in view of the implications for missing significant pathology.

Our study also shows that the cold caloric test has a poor sensitivity compared to Bithermal caloric test and so is not a useful test to screen patients with vertigo with vestibular pathology. Even though the specificity of cold caloric test is 83% it may still miss those patients with significant pathology by this test alone

A questionnaire takes about 40 minutes and identifies the aetiology while Bithermal caloric test takes 100 minutes and costs Rs 600 per test in CMC and measures the function of the labyrinth and its location and the side of the lesion. These being complementary, are both needed for final management and follow up of the patient.

This study shows that the cold caloric test has a poor sensitivity compared to Bithermal caloric test and so is not a useful test to screen patients with vertigo with vestibular pathology. If cold caloric test is used to rule out patients with

pathology, a specificity of 83% is not adequate since one would like a test with close to a specificity of 100% for ruling out a pathology.

Based on these findings we recommend that all patients coming to the clinic are evaluated using the structured questionnaire, followed by otoneurological evaluation.

CONCLUSION

In tertiary care, all patients with giddiness should be screened with the questionnaire and those identified as symptomatics require Bithermal caloric testing to identify the site and side of the lesion before specific treatment is started. The use of cold caloric test as a screening test should be discontinued forthwith.

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ANNEXURE – I

Vertigo clinic evaluation form

Name **Age** **Sex** **Hospital no** **Address**

Presenting complaint: vertigo /lightheadedness / imbalance /others_____

1. VERTIGO (An illusion of movement (Rotatory /linear displacement)

Duration of vertigo hours days months years

Type of vertigo: episodic continuous uncertain

Present at rest at rest and movement on movement only

Head rotating surrounding rotating uncertain

Description of first attack: same as present attacks yes/no different from present attacks
How? ----------

Description of a typical attack:

Duration of each attack seconds minutes hours days
weeks (Max Min)

Date of last attack **Frequency of each attack**

Periods in between attacks free of symptoms not free of symptoms

Warning signs before attack fullness in ear/aura/ others /nil

Clustering of attacks yes no

Aggravating/precipitating factors: nil/coughing/sneezing/loud sounds/specific food/ specific head positions
Standing from sitting position/turning in bed /raising hands/ others

Relieving factors nil/yes (specify_____)

Vegetative symptoms (nausea/vomiting/sweating/others) yes no

Positional vertigo yes no **drop attacks** yes no **Oscillopsia** yes no

Motion sickness yes no **Acoustic trauma** yes no

Difficulty walking in the dark/streets/open spaces yes no

Any URI/fever before attack yes no **Any barotrauma** (swim/fly) yes no

Fullness/pressure sensation in the ear yes no

Tinnitus rt (yes/no) left (yes/no) annoyance / continuous / intermittent

hearing loss rt(yes/no) left(yes/no) fluctuating (rt/left)/nil phonophobia; better/ worse in noisy surroundings/ rapidly progressing hearing loss/ sudden in onset

Other ENT complaints no yes (ear discharge ,others)

Neurological complaints no yes (specify dysarthria / diplopia/ headache/ loc / blackouts / pins and needles or tingles in hand and feet / facial pain or numbness / spots before eyes/ seizures / others)

Headache: no yes severity side/site associatednausea/vomiting/scotomas/aura/others

Cervical pain no yes **loss of balance on walking** no yes

Cardiovascular disorders no yes (past h/o MI/palpitations/ chest pain/leg pain on walking or at rest /ccf/others)

Medical problems no yes (thyroid / DM / anemia /polycythemia/ autoimmune/ TB / smoking /alcohol / /loss of wt / appetite / blood in stools /diarrhea / food intolerance/ indigestion / bleeding disorders /macroglobinaemia/ others)

Eye problems no yes(loss of vision /pain /discharge or tearing / glaucoma/diplopia/refractory errors/new glasses/others)

Head injury/ any trauma no yes ( )

Ototoxic drugs/other medications no yes (Immunosupressents/steroids/others)

Medicines taken/ taking (and duration)

Psychiatric no yes (Insomnia / depression / conversion reactions/agrophobia/others)

Any known allergy no yes( )

Family h/o giddiness/Psych dis no yes( )

Time off work/school

2. **NEAR FAINT**-Sensation of impending faint (light headedness) ***mechanism***-Diffuse cerebral ischemia (tunneling /dimming of vision, shortness of breath, air hunger, perioral numbness)

Types

Orthostatic hypotension- reduced blood volume, hypotensive drugs, autonomic dysfunction

Vasovagal attack- prolonged standing in hot sun, fear**,** severe pain, acute vertigo

Hyperventilation- anxiety, stress, panic attacks

Decreased cardiac output –arrhythmia, valvular disease, heart failure

If 2 H/so orthostatic hypotension

Postural hypotension no yes **on any hypotensive drugs** no yes

Antidepressants no yes major tranquilizers no yes

Any h/o autonomic dysfunction no yes
(Bladder / bowel dysfunction/ periph neuropathy)

H/so vasovagal attack no yes
(Associated with prolonged standing/ severe pain/ severe vertigo preceding) no yes
H/so hyperventilation no yes
(anxiety/stress/panic attacks /associated symptom -frequent sighing, air hunger/tightness in chest/ perioral numbness/paresthesia of extremities)

H/so reduced cardiac output no yes
(Arrhythmia / valvular disease/heart failure)

3. PSYCHOPHYSIOLOGICAL DIZZINESS - Sensation of floating/swimming /rocking/spinning inside head (not associated with an illusion of movement of environment)
mechanism– impaired central integration of sensory signals

If 3. Constant in attacks
? Associated with tension headache /palpitations / fatigue / weakness no yes

? Precipitating factors social situations / driving in open spaces no yes

4. **MULTISENSORY DIZZINESS** ***mechanism–***partial loss of multiple sensory system function

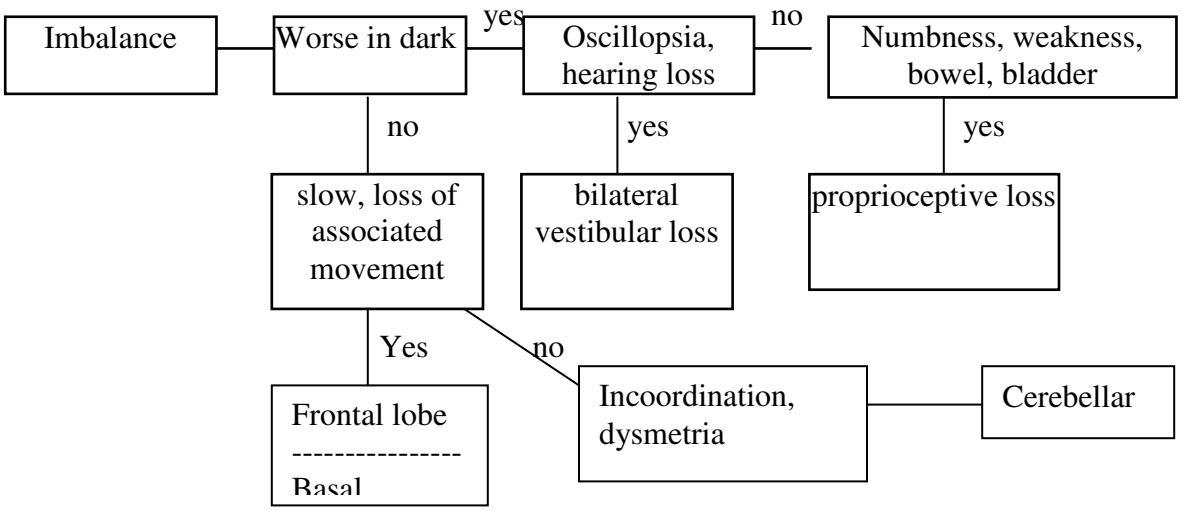
Any systemic diseases ***no/yes(peripheral neuropathy/decreased visual acuity/hearing impairment/vestibular impairment sec to ototoxic drugs/others)***

5. PHYSIOLOGICAL OVERLOAD
mechanism- sensory conflict due to unusual combination of sensory signals

Any h/o air travel/sea travel no yes H/o motion sickness no yes

6. IMBALANCE / DYSEQUILIBRIUM-(only on standing/walking and not related to abnormal head sensation) ***mechanism-*** loss of vestibulospinal, proprioceptive, cerebellar, or motor function

Imbalance/dysequilibrium (algorithm for localizing the lesion) fig 1



If 4.

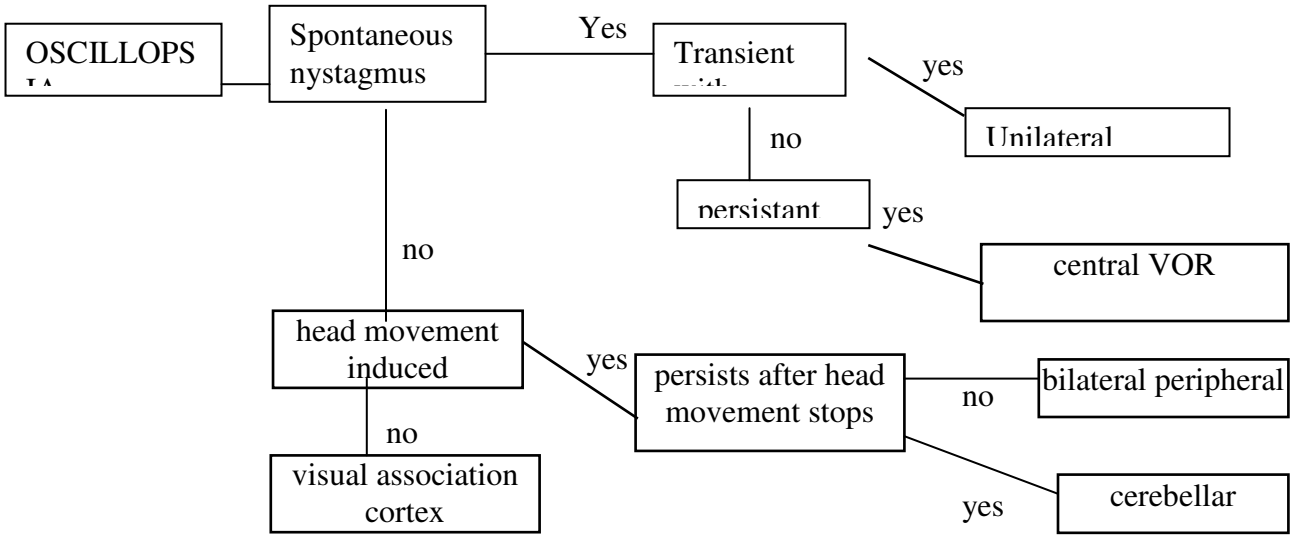
Worse in dark no yes oscillopsia no yes

Numbness / weakness / bladder bowel dysfunction no yes Slow loss of associated no yes

Movement dysfunction; incordination / dysarthia no yes

5. **OCULAR DIZZINESS *mechanism*-** visual-vestibular mismatch due to impaired vision

Algorithm for localizing lesions in patients complaining of oscillopsia (fig 2)



NEURO-OTOLOGICAL EXAMINATION

Systemic **Pallor** y / n **BP** lying standing

Bruits carotids no / yes rt / lft

Peripheral pulses dorsalis rt/lft radial rt/lft

Ears **TM** rt lft

TFT **Rennie** rt lft **Webbers** rt lft **ABC** rt lft

Cranial nerves **corneal** rt lft

Eye movements normal /abnormal **ptosis** no yes

7th **9th** **10th** **11th** **12th**

Neurological system **deep tendon reflexes** normal /abnormal **Babinsky** normal /abnormal

muscle strength normal /abnormal **sensation** -**face** normal /abnormal **limbs** normal /abnormal

Cerebellar functions **Finger to nose** eyes openeyes closed

EXAMINATION OF BALANCE SYSTEM

1) VESTIBULOSPINAL

STATIC IMBALANCE

Ronberg's **standing test** **Walking-tandom**

eyes open *eyes closed* *eyes open* *eyes closed* *eyes open* *eyes closed*

Untenbergers test *Fukadas writing test eyes open* *eyes closed*

DYNAMIC VESTIBULO-SPINAL FUNCTION

EXAMINATION OF GAIT

2)VESTIBULO-OCCULAR SYSTEM

ABNORMAL EYE MOVEMENTS

Opsoclonus yes no **ocular bobbing** yes no **ocular flutter** yes no

Ocular myoclonus yes no

Inspection of spontaneous nystagmus

Using Frenzel Lens no/yes [horiz(rt/ lft) rotat(clockwise/anticlw) vert (up/dn)
(Without optic fixation) deg 1 2 3 deg 1 2 3 deg 1 2 3
jerky/pendular direction changing /direction fixed

Without Frenzel lens no/yes horiz(rt/ lft) rotat(clockwise/anticlw) vert(up/dn)
(with optic fixation) deg 1 2 3 deg 1 2 3 deg 1 2 3

Gaze-holding nystagmus horiz no yes (rt /lft)

Skew deviation and ocular tilt reaction

The alternate cover test/Madrox rod **horiz** no / yes **vertical** no / yes

Saccades no / yes

Smooth pursuit normal / abnormal {corrective saccades no yes (number)}

Head shaking nystagmus no yes (horizontal rt/left vertical no / yes)

Head -thrust test ***no*** ***yes rt / lft***

Positioning testing ***Dix- Hallpike maneuver***

.

Positional nystagmus sitting HS HR HL HE HF HP
supine HS HR HL HE HF HP

Dynamic visual nystagmus no yes **Valsalva induced nystagmus** no yes

Hyperventilation **dizziness** no yes **nystagmus** no yes

Tullio phenomenon no yes **Fistula test** no yes

Vertigo clinic evaluation form

Name Age Sex Hospital no Address

DIAGNOSIS AFTER HISTORY AND OTONEUROLOGICAL EXAMINATION

1)_____ 2)_____3)_____

INVESTIGATION ASKED FOR AND RESULTS

AUDIO PTAright_____left_____

IMPEDENCE right_____left_____SPEECH TESTS right_____left_____

ENG PENDULAR SPONTANEOUS NYSTAGMUS

GAZE NYSTAGMUS

CALORIC

BLOODS_____

IMAGING_____

GLYCEROL TEST right_____left_____ **OTHERS**_____

REFERAL ASKED FOR AND THEIR IMPRESSION

_____

_____

_____

VERTIGO SYMPTOM SCALE **vertigo severity_____somatic anxiety_____**

FINAL DIAGNOSIS_____

_____

MANAGEMENT PLAN

Medication_____

Vestibular exercises **custom made _____** **routine_____**

surgery

FOLLOW UP **3months** **6 months**
1 yr **2yr**

ANNEXURE – II

Annexure 2
Consent form for the study proposal' Validity of Minimal Cold Irrigation test (MCIT) used in tertiary care for diagnosing labyrinthine derangement'

CONSENT FORM

Title of project: Validity of Minimal Cold Irrigation test (MCIT) used in tertiary care for diagnosing labyrinthine derangement.

Name of Researcher: Dr Ramesh Menon

PLEASE INITIAL BOX

1. I confirm that I have read and understood the information sheet dated …….. for the above study and have had the opportunity to ask questions. ☐
2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. ☐
3. I understand that sections of any of my medical notes may be looked at by responsible individuals from (company name) or from regulatory authorities where it is relevant to my taking part in research. I give permission for these individuals to have access to my records. ☐
4. I agree to take part in the above study. ☐

_____	_____	_____
Name of patient	Date	Signature
_____	_____	_____
Name of Person taking consent (if different from researcher)	Date	Signature
_____	_____	_____
Researcher	Date	Signature

Master chart: Controls

PATIENT	Hosp_ no	case2 contr1	Age	Sex	Dur_rt (secs)	Nor 1 Abn 2	Dur _lft	Nor 1 abn2	VP freq	Nor 1 Abn 2	VP Spv	Nor 1 Abn 2	DP Freq	Nor 1 Abn 2	DP Spv	Nor 1 Abn 2	Overall Nor 1 Abn 2
SHREVA	485946c	1	23	f	150	1	130	1	4.1	1	5.2	1	0.8	1	2.7	1	1
SUDIN	903272B	1	25	m	105	1	130	1	16.5	1	5.4	1	4.9	1	33.3	1	1
SINDU	321050c	1	20	f	110	1	90	1	4.8	1	13.7	1	18.1	1	4.8	1	1
INDULEKA	321052c	1	22	f	100	1	100	1	13.9	1	22.6	2	11.5	1	8	1	2
DEVA	864989c	1	23	m	90	1	75	1	12.2	1	12.7	1	10.8	1	5	1	1
MALINI		1	28	f	70	1	70	1	10.3	1	3	1	4.4	1	24.1	1	1
NARENDRA	947348c	1	29	m	90	1	110	1	27.5	2	6.4	1	2.6	1	5.8	1	2
MERCY	442876a	1	21	f	110	1	100	1	6	1	5.6	1	8.4	1	24.1	1	1
RANJIT	299834c	1	49	m	110	1	120	1	1.2	1	5.1	1	7	1	11.1	1	1
HANNA	480238c	1	19	f	130	1	120	1	6.2	1	6.4	1	1.2	1	4.4	1	1
SOMU	664301c	1	18	f	90	1	100	1	4.3	1	12.8	1	1.4	1	13.8	1	1
SUSAN	321001c	1	21	f	90	1	80	1	1.9	1	19.6	1	2.8	1	12.6	1	1
SHARMILA	584812a	1	21	f	155	1	165	2	0	1	3.7	1	2.4	1	16.4	1	1
VENKATESH		1	47	m	120	1	120	1	10.2	1	8.8	1	22.6	1	16.6	1	1
SNEHA	042072c	1	27	f	130	1	110	1	2.2	1	7.2	1	8.7	1	24.7	1	1
BAMINI	632521c	1	18	f	75	1	75	1	11.6	1	2.4	1	2.4	1	11.5	1	1
JANSI	044925c	1	30	f	70	1	70	1	2.8	1	6.5	1	2.8	1	14.9	1	1
JANSI RANI	864977c	1	21	f	130	1	130	1	1.7	1	0.5	1	8.5	1	0.2	1	1
KULASEKARAN	951946c	1	22	m	85	1	100	1	7.1	1	16.3	1	4.5	1	15.5	1	1
ARUN		1	36	m	100	1	130	1	13.9	1	2.3	1	3	1	2.8	1	1
NEELDOSS	780165	1	29	m	80	1	100	1	11.9	1	24.7	1	13.1	1	24.5	1	1
AMSAVALLI	190195c	1	26	f	120	1	130	1	0	1	6.4	1	12.1	1	9.5	1	1
SRINIVASAN		1	56	m	60	1	130	1	15	1	7.9	1	0.4	1	16.1	1	1
JONSY	321013c	1	23	f	130	1	110	1	5	1	14.3	1	24	1	25	1	1
PRITIV JOEL	485922c	1	20	m	90	1	100	1	2.9	1	3.9	1	6.4	1	4.4	1	1

SOHINI	485802c	1	22	m	80	1	80	1	14.3	1	7.3	1	6.3	1	1.6	1	1
PREETIV	485898c	1	22	m	60	1	100	1	23.2	2	31.2	2	13	1	35.9	2	2
JOJIN	178774c	1	23	m	65	1	55	1	4.8	1	8.2	1	4.8	1	4.4	1	1
RAMA DEVI	485821c	1	22	m	150	1	85	1	9.3	1	11.4	1	6.6	1	6.2	1	1
KULAKESARAN	951946c	1	22	m	75	1	80	1	3.9	1	4.2	1	5.8	1	5.5	1	1

Master Chart: Symptomatics

PATIENT	Hosp_no	case2 contr1	AGE	SEX	Dur_rt (secs)	Nor 1 Abn 2	Dur _lft (sec)	Nor 1 abn2	VP fre (% of)	Nor 1 Abn 2	VP Spv (%)	Nor 1 Abn 2	DP Freq (%)	Nor 1 Abn 2	DP Spv (%)	Nor 1 Abn 2	Overall Nor 1 Abn 2
santhan	038576c	2	38	m	90	1	90	1	4.2	1	30.5	2	9.7	1	41.8	2	2
arvind	026516d	2	34	m	70	1	70	1	7.4	1	2.4	1	2.5	1	12.6	1	1
kanniapp	046755b	2	60	m	60	1	60	1	19.7	1	25	1	36.8	2	35	2	2
jothi	010443b	2	35	f	100	1	90	1	36.9	2	42.2	2	18.5	1	37.6	2	2
lalitha	154214b	2	45	f	80	1	80	1	22.5	2	41.8	2	0.9	1	13.5	1	2
archana	035490c	2	37	f	70	1	70	1	20.9	1	46.8	2	14.9	1	1.6	1	2
dilip	998760c	2	35	m	90	1	90	1	14.1	1	44.4	2	10.9	1	35.2	2	2
rangaswa	998760b	2	60	m	130	1	130	1	48.6	2	33.5	2	6.8	1	33.5	2	2
rajeswari	584796c	2	45	f	120	1	120	1	17.6	1	23.9	2	12.2	1	26.7	1	2
sudershan	994830c	2	55	m	90	1	90	1	6	1	11.2	1	3	1	3.9	1	1
nagarat	589174c	2	45	f	90	1	95	1	9.7	1	5.5	1	16.7	1	11.7	1	1
dina	935309c	2	33	f	70	1	80	1	5.3	1	0.7	1	15.3	1	3.1	1	1
geeta	898389b	2	27	f	75	1	100	1	10.5	1	29.7	2	7	1	18.9	1	2
gyani	032818d	2	45	m	15	2	90	1	3.2	1	17.4	1	13.7	1	3	1	1
uma	034216d	2	38	m	95	1	90	1	18.6	1	28.1	2	10.3	1	24.4	1	2
Dhanajay	018938d	2	37	m	70	1	60	1	6.7	1	56.6	2	15.6	1	1.9	1	2
md ali	041501d	2	32	m	100	1	75	1	26.6	2	41	1	25	1	2.1	1	2
sitaram	044360d	2	55	m	100	1	90	1	37.7	2	55.6	2	10.7	1	23.5	1	2
iqbal	042402d	2	37	m	50	1	30	1	1.1	1	9.6	1	3.4	1	48.9	2	2
krishna L	102878d	2	43	m	110	1	80	1	15.8	1	26.4	2	68.4	2	58.2	2	2
anil chandra	117867d	2	57	m	40	2	90	1	18.5	1	51.7	2	0.6	1	12.3	1	2
Gomathi	878208c	2	23	f	35	2	25	2	11.1	1	44.5	2	16.7	1	24.9	1	2
Amit	061473d	2	17	m	90	1	90	1	9.3	1	34.4	2	0.6	1	6.3	1	2
manju g	116586d	2	27	f	90	1	100	1	8.3	1	11.9	1	28.7	2	28.4	1	2
susanta	000513d	2	33	f	70	1	0	1	2.7	1	8.7	1	2.7	1	16.8	1	1
anil c	117867d	2	67	m	25	2	70	1	18.5	1	51.7	2	0.6	1	12.3	1	2
champa roy	122160d	2	65	m	100	1	95	1	19.6	1	33.4	2	1.5	1	24.5	1	2
nageswar	024161d	2	52	m	180	1	120	1	18.6	1	20.8	1	1.2	1	47.1	2	2
sabitha	721782c	2	50	f	115	1	100	1	4.4	1	0.5	1	25.1	2	18.9	1	2

rina	039802d	2	35	f	100	1	80	1	4.8	1	18.8	1	29.5	2	23.3	1	2
santhan	038576c	2	38	m	90	1	90	1	4.2	1	30.5	2	9.7	1	41.8	2	2
arvind	026516d	2	34	m	70	1	70	1	7.4	1	2.4	1	2.5	1	12.6	1	1
kanniapp	046755b	2	60	m	60	1	60	1	19.7	1	25	1	36.8	2	35	2	2
jothi	010443b	2	35	f	100	1	90	1	36.9	2	42.2	2	18.5	1	37.6	2	2
lalitha	154214b	2	45	f	80	1	80	1	22.5	2	41.8	2	0.9	1	13.5	1	2
archana	035490c	2	37	f	70	1	70	1	20.9	1	46.8	2	14.9	1	1.6	1	2
dilip	998760c	2	35	m	90	1	90	1	14.1	1	44.4	2	10.9	1	35.2	2	2
rangaswa	998760b	2	60	m	130	1	130	1	48.6	2	33.5	2	6.8	1	33.5	2	2
rajeswari	584796c	2	45	f	120	1	120	1	17.6	1	23.9	2	12.2	1	26.7	1	2
sudershan	994830c	2	55	m	90	1	90	1	6	1	11.2	1	3	1	3.9	1	1
nagarat	589174c	2	45	f	90	1	95	1	9.7	1	5.5	1	16.7	1	11.7	1	1